and termination were exceedingly like acute myelitis, but in which a careful microscopic examination showed entire absence of inflammatory changes, the sole lesion being an endarteritis of the pial vessels. The duration of the disease from the appearance of distinct spinal symptoms to the fatal termination was from eight to twenty days, and, as stated, the clinical picture was that of acute myelitis; that is, quickly progressing paraplegia with sensory disturbance, paralysis of bladder and bowel and the rapid formation of massive decubitus.

The author is at great pains to demonstrate what has long been known to most neurologists; viz., that there is such a thing as softening of the cord from vascular occlusion, and that it is quite distinct from myelitis.

The obliterating endarteritis in the reported case was quite sufficient to produce all the symptoms noted, and the principal points of interest are: first, the very slight changes discovered in the nerve fibres and cells of the cord; second, the occurrence in one case of the sensory dissociation generally found in syringomyelia and almost peculiar to it (i. e., loss of pain and temperature sense with preservation of tactile sense); third, the peculiar relation of the knee-jerks to the other symptoms and the seat of the most advanced lesions; and fourth, lack of explanation of the rapid course of the disease, the arterial changes being essentially chronic in character. The last difficulty has apparently not occurred to the author, but would seem to demand elucidation. The almost normal appearance of the cord was probably due to the short course of the disease, sufficient time not having elapsed for the occurrence of chemical changes in the tissues that we know as disintegration and degeneration. Regarding the condition of sensation and the apparent inconstant behavior of the deep reflexes, it can only be said that there is much still to be learned concerning this, and cases of diffuse lesion such as those reported by Biernacki, are illy adapted for illumination on these subjects

On the whole, we see no adequate reason why these three cases should constitute an excuse for adding a new term to neuropathology. Arterio-sclerosis of the spinal arteries, due to syphilis, senility and other causes, is well known, sufficiently comprehensive and sufficiently exact to embrace all such cases, but we do think that it should be more constantly in the mind of the practitioner than is at present the case. PATRICK.

265. KLINISCHER BEITRAG ZUR LEHRE VON DER DYSTROPHIA MUSCULARIS PROGRESSIVA **(A Clinical Contribution to Progressive Muscular Dystrophy). J. Hoffmann (Deutsche Zeitschrift für Nervenheilkunde, 12, 1897-1898, p. 418).**

Hoffmann reports two cases in twin brothers which show that progressive muscular dystrophy may appear in the clinical picture of bulbar paralysis, contrary to the generally accepted opinion. The imperfect closure of the eyelids was noticed by the mother in both boys in early infancy, and this indicated that the disease was congenital or early acquired. Later, paralysis and atrophy of the facial muscles, atrophy of the tongue, paralysis of the soft palate—in one child paralysis of the muscles of mastication—were observed. The disease differed from bulbar paralysis in the involvement of the muscles of the forehead, and in the absence of fibrillary twitching and reaction of degeneration; and differed from the family form of infantile bulbar paralysis in the mask-like expression of the face, the tapir mouth, the lagophthalmos, the condition of the muscles elsewhere in the body, etc.

As differential points between the family form of infantile progressive bulbar paralysis (Fazio, Londe) and the bulbar form of muscular dystrophy, Hoffmann mentions the rapid progress of the former disease, as well as the fibrillary twitching, the reaction of degeneration, changes in other nerves (opticus), and the absence of the ordinary signs of dystrophy in the trunk and limbs. In some cases the diagnosis is impossible.

Between the congenital absence of the nuclei and the dystrophy of the facial muscles, the arrest of the process is a sign in favor of the former condition, especially when the process is unilateral and combined with abducens paralysis. In one of Duchenne's cases of muscular dystrophy, however, the atrophy of the muscles was confined to the face for thirty years. The occurrence of the atrophy in more than one member of the family and the tapir mouth are in favor of the diagnosis of dystrophy.

Hoffmann reports two cases which show that in muscular dystrophy the atrophy may begin in the peripheral parts of the limbs, and the condition may resemble that of progressive neurotic muscular atrophy.

Hoffmann shows in this paper that muscular dystrophy (1) may begin as bulbar paralysis and remain as such a long time; (2) that it may begin in the lower leg and forearm muscles, and (3) that external ophthalmoplegia may be one of its signs. All the voluntary muscles may be affected in muscular dystrophy, but no case has *yet* been reported in which the disease began in the small muscles of hand and foot. He is inclined to class the much-cited case of Oppenheim and Cassirer under the dystrophies, and not to regard it as a special form of disease. SPILLER.

266. UEBER CENTRALE ERWEICHUNG DES RUECKENMARKES BEI MENINGITIS SYPHILITICA (On Central Softening of the Spinal Cord in Syphilitic Meningitis). Wullensoeber (Münchener Medicinische Wochenschrift, 45, 1898, p. 1,017).

A woman of 28 years, with a history of having had syphilis, presented the following train of symptoms: first, severe pains in the loins radiating to the front of the abdomen, next headache, then weakness and tonic contractions in the legs; patellar reflexes were lost, and she had intercurrent disturbances of vision, but no alteration of sensation for either touch, pain or temperature, and no ataxia. The trouble progressed, there was complete paralysis of the legs, with atrophy of the muscles, and sphincter paralysis, bed sores developed. The patient died about fifteen months after coming under observation. The autopsy showed syphilitic cerebro-spinal meningitis, with compression of the spinal cord, and the production of a central cavity extending from the midlumbar to the upper dorsal region of the cord. This cavity was most developed in the middle and lower dorsal region, where it occupied almost the whole of the usual site of the gray matter. No development of gliomatous tissue was to be found about the borders of the cavity. There was ascending degeneration in the cervical, and descending degeneration in the lower lumbar region. The arteries of both brain and cord showed characteristic syphilitic arteritis. On account of the absence of gliomatous tissue, the author thinks that syringomyelia can be excluded, and regards the case as one of necrosis of the spinal cord, due to syphilitic disease of the vessels. On searching the literature of the last twenty years he could find but four observations of cases in which syphilitic meningitis was accompanied by the formation of a cavity in the spinal cord.

ALLEN.